

Table 1 Distribution of Cuban PPNG and non-PPNG strains from 1995 to 1998

Year	No of gonococci examined	PPNG strains		Non-PPNG strains	
		No	%	No	%
1995	63	33	52.4	30	47.6
1996	21	14	66.6	7	33.4
1997	21	13	61.9	8	38.1
1998	5	1	20	4	80
Total	110	61	55.5	49	44.5

PPNG = penicillinase producing *N. gonorrhoeae*.

have been recently evaluated in Cuba with good results (R Llanes, *et al*, unpublished data, 1999).

We thank Lic D Guzman, Lic Y Gutierrez, and O Gutierrez for their technical support during this study and Dr A Llop for her revision.

R LLANES

J SOSA

I MARTINEZ

National Reference Laboratory for Neisseria, Tropical Medicine Institute "Pedro Kouri" (IPK), PO Box 601, Havana, Cuba

Correspondence to: Dr Rafael Llanes Caballero

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Accepted for publication 5 November 1999

Rising HIV prevalence in STD clinic attenders at Chandigarh (north India)—a relatively low prevalence area

EDITOR,—The patients attending the STD clinics are at risk of having concurrent HIV infection. The trends of HIV infection in these patients may reflect the trends of HIV epidemic in the community. We have analysed the HIV status of 981 patients (824 males, 157 females) who attended our STD clinic from January

1993 to July 1999 (about 6½ years). The screening for HIV was done by ELISA. Those who were found positive were tested by repeat ELISA utilising another blood sample and considered HIV seropositive only, if both samples were found positive. The STDs were diagnosed by appropriate laboratory tests. The majority of the attenders had STDs; however, a small but significant proportion of patients had psychosexual disorders and other non-sexually transmitted genital diseases. Four per cent of the 981 patients—that is, 40 patients (26 males, 14 females) were found to be seropositive for HIV. The annual prevalence showed a rising trend (1993, 0.56%; 1994, 4.4%; 1995, 2.4%; 1996, 4%; 1997, 4.4%; 1998, 5.7%; and January to July 1999, 8.7%). The prevalence of HIV seropositivity in different STDs is shown in table 1. Large proportions of seropositive patients were truckers (15/40, 37.5%) and housewives (12/40, 30%). Among 12 housewives, four were wives of truckers. All of the 26 seropositive male patients confessed to at least one sexual contact with commercial sex workers (CSWs). Twenty eight (70%) seropositive patients had one STD, while the remaining 12 (30%) patients had more than one STD; 18 (45%) seropositive patients had STDs with either atypical morphologies or unusual severity, the remaining 22 (55%) presented with usual morphologies.

India is a country with a wide variation in geographical, cultural, and behavioural patterns. This is also reflected in the trends of current HIV epidemic in the various regions of the country. We believe that no other country has such a high intranation variation in HIV epidemic status. Comparison of our data on HIV prevalence with STD clinics of different regions of the country highlights this difference. The high HIV prevalence zones of the country include western and southern zones, where HIV prevalence among STD clinic attenders varies from 15% to 33%.¹⁻³ On the other hand, in eastern and northern zones, it is still low and varies from 0.2 to 4%.¹⁻³⁻⁵

In our study we found that a high proportion of HIV positive patients were truckers, who generally acquired infection from CSWs from the highways to Bombay or Chennai, two metropolitan cities of the western and southern zones respectively. These long distance truckers have a high risk sexual behaviour and contribute in the spread of HIV infection throughout the country in a short time.²⁻⁶

Even though the present figures for HIV seropositivity in STD clinic attenders are not very high, the HIV epidemic in this region is now progressing at an alarming rate. In our

study, the prevalence in our STD clinic increased from 0.56% in 1993 to 8.7% in 1999 (to July). This indicates that northern India is entering from a low level epidemic (HIV prevalence less than 5% in STD patients) to a concentrated epidemic.¹ This calls for an immediate vigorous intervention programme to be introduced in this region.

BHUSHAN KUMAR
SOMESH GUPTA

Department of Dermatology, Venereology and Leprology, Postgraduate Institute of Medical Education and Research, Chandigarh-160 012, India

Correspondence to: Dr Kumar

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Accepted for publication 5 November 1999

HIV seropositivity in women with syphilis in Delhi, India

EDITOR,—There has been a progressive rise in the prevalence of human immunodeficiency virus (HIV) infection in India, which currently has the largest number of HIV infected people in the world.¹ The spread of HIV is predominantly by heterosexual transmission in India.² Sexually transmitted disease (STD), particularly genital ulcer disease (herpes, syphilis, and chancroid), has an important role in the transmission of HIV, and the two have been observed to be interrelated.³⁻⁴ We conducted a pilot study to assess the relation between syphilis and HIV infection among non-pregnant women attending gynaecology and STD clinics of our hospital.

From June 1998 to July 1999, sera from 281 non-pregnant women were tested for syphilis by VDRL (Serologist, India) and confirmed by TPHA (Immunotep, Omega Diagnostic Ltd, UK). Sera that tested positive for syphilis were tested for HIV without identifying the patient. Individual informed consent for HIV was not obtained as results were not aimed to be linked to the identity of those tested. Serum was tested first with one ELISA/rapid/simple (ERS) assay, utilising either of the three different enzyme linked immunosorbent assay (UBI, HIV-1/2, United Medical Inc, USA, Recombigens HIV-1/HIV-2, EIA, Cambridge Biotech Galway, Ireland, and HIV spot Genelabs Diagnostic, Singapore). Any reactive sample was retested using a different assay. Samples that were reactive in all the three tests were considered HIV antibody positive. A sample that was non-reactive on the first test was considered HIV negative, as was a sample that was reactive in the first and non-reactive in the next test.⁵

Of 281 sera tested, 48 (17%) were seropositive for syphilis. HIV antibody was detected in sera of six (12.5%) patients who were seropositive for syphilis (table 1). None of the 233 patients with negative syphilis serology tested

Table 1 Frequency of HIV seropositivity in different sexually transmitted diseases

STDs	No screened	HIV seropositive	Seropositivity rate (%)
Ulcerative STDs			
Genital herpes	188	19	10.1
Syphilis	107	6	5.6
Chancroid	21	1	4.76
Donovanosis	5	0	0
Lymphogranuloma venereum	5	0	0
All ulcerative STDs	322	25	7.6
Non-ulcerative STDs			
Condyloma acuminata	184	13	7
Balanoposthitis	75	2	2.66
Gonorrhoea	35	1	2.85
Molluscum contagiosum	27	3	11.1
Non-gonococcal urethritis	27	0	0
Vaginitis	23	1	4.3
All non-ulcerative STDs	368	18	4.9
All STD clinic attendees*	981	40	4

*The discrepancy in total is due to the presence of more than one STD in some patients.

Table 1 Details of patients undergoing serological test for syphilis

Clinical diagnosis	No of samples (%)	Positive for syphilis serology	Positive for HIV
Previous pregnancy loss*	89/281 (31.6)	16/89 (17.9%)	0/16 (0%)
Vaginal discharge	101/281 (55.8)	9/101 (8.9%)	1/9 (11.1%)
Genital growth	49/281 (17.4)	6/49 (12.2%)	1/6 (16.6%)
Genital ulcer	42/281 (14.9)	17/42 (40.47%)	4/17 (23.5%)

*Intrauterine death, still birth, repeated abortions.

positive for HIV antibody. This was highly significant ($p < 0.001$, Fisher's exact test). Presence of HIV antibody was associated with genital ulcer in 23.5% women, followed by genital growth and vaginal discharge in 16.6% and 11.1% respectively.

There is a higher prevalence of STD and HIV infection among men compared with women. HIV seropositivity has been associated with a reactive serological test for syphilis among males. This could be probably due to higher percentage of male attendance in STD clinics.⁶ We therefore undertook this study to evaluate if some association exists between syphilis and HIV among non-pregnant women attending the gynaecology clinic, as well as the STD clinic. Untreated STDs, especially those with ulcerative disease, can enhance both susceptibility of a person to HIV infection as well as infectivity of HIV positive individual. Breach in the epithelial surface of a genital ulcer may be an important factor in the transmissibility of HIV. This is evident from our results where incidence of positive serology for HIV was highest among women with genital ulcer (23.5%). Our study demonstrates a significant association between positive serology for syphilis and presence of HIV infection. We feel that the diagnosis of syphilis in non-pregnant women may act as a marker to detect the presence of HIV infection.

M VAJPAYEE

Department of Microbiology, All India Institute of Medical Sciences, New Delhi, India

N MALHOTRA

Department of Obstetrics and Gynaecology

P SETH

Department of Microbiology

D TAKKAR

Department of Obstetrics and Gynaecology

R M PANDEY

Department of Biostatistics

Correspondence to: Madhu Vajpayee, MD, Department of Microbiology, All India Institute of Medical Sciences, New Delhi-110029, India

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Accepted for publication 23 November 1999

Immune reconstitution CMV pneumonitis

EDITOR,—A 41 year old white homosexual man presented in late July 1999 with a 5 day history of exertional dyspnoea, non-productive cough, fever with sweats, and anorexia. An empirical course of broad spectrum antibiotics did not improve his symptoms and SaO_2 remained $\leq 95\%$ on air at rest. The chest radiograph showed non-specific abnormalities. He had been found to be HIV-1 antibody positive in August 1991; cutaneous Kaposi's sarcoma defined AIDS in June 1992. In May 1995 biopsy confirmed cytomegalovirus (CMV) oesophagitis and colitis were treated with intravenous ganciclovir for 2 weeks; no maintenance therapy was given. At this time the CD4 count was 130 cells $\times 10^6/l$. In October 1996 the patient had *Pseudomonas aeruginosa* pneumonia. He had a complex antiretroviral history, having taken combinations of reverse transcriptase inhibitors and protease inhibitors. He had discontinued all antiretroviral therapy in January 1999 as therapy had failed to maintain CD4 counts and HIV viral load had risen: co-trimoxazole primary *Pneumocystis carinii* pneumonia prophylaxis had been continued. In early June 1999 HIV viral load had risen to 223 000 copies/ml and CD4 count had fallen to 70 cells $\times 10^6/l$. Two weeks before the onset of respiratory symptoms the patient had recommenced antiretroviral therapy with d4T, 3TC, and amprenavir/saquinavir. Four weeks after starting antiretroviral therapy viral load had fallen to 1500 copies/ml and CD4 had risen to 170 cells $\times 10^6/\mu l$. A computed tomography (CT) scan of the thorax 4 weeks after the onset of respiratory symptoms and 6 weeks after starting antiretroviral therapy showed focal areas of ground glass shadowing, largely in the left upper lobe but also involving other lobes; in addition, chronic changes resulting from the previous episode of pneumonia were noted, including multifocal fibrotic change with thickened interlobular septae, cystic air spaces, and minor bronchiectasis involving all lobes. Repeat viral load at this time = 200 copies/ml and CD4 = 160 cells $\times 10^6/l$. At bronchoscopy, performed after 8 weeks of antiretroviral therapy, the endobronchial appearances were normal. Bronchoalveolar lavage (BAL) was performed from the left upper lobe. Analysis of BAL fluid revealed a lymphocytic reaction; many cells had intranuclear/cytoplasmic inclusions typical of CMV infection. In situ hybridisation for CMV was positive. Staining and culture for bacteria, mycobacteria, *P carinii* and other fungi were negative. Intravenous ganciclovir 10 mg/kg per day was given for 21 days, in addition, antiretroviral therapy and co-trimoxazole were continued. With this there was a rapid defervescence of fever, a reduction in exertional dyspnoea and improvement in SaO_2 to $\geq 98\%$ on air. Repeat CT of the thorax after 3 weeks of intravenous ganciclovir showed an improvement in ground glass shadowing and persistence of the chronic

changes. The patient was subsequently maintained on oral ganciclovir.

The diagnosis of CMV pneumonitis was made by identifying CMV as the sole pathogen in BAL fluid and the improvement in symptoms, SaO_2 , and CT appearances with ganciclovir as monotherapy. This diagnosis was made in the context of a rapidly falling viral load and an increase in CD4 count indicating partial immune reconstitution.

Partial restoration of cell mediated immunity induced by antiretroviral therapy, as shown by recovery of part of CD4 T cell reactivity to memory antigens,^{1,2} may cause development of sufficient inflammatory responses to produce symptoms and signs in patients latently infected with opportunistic infections. Reactivation mycobacterial lymphadenitis,³ cryptococcal meningitis,⁴ and CMV retinitis^{5,6} have been described. The case described here suggests CMV pneumonitis should be added to the list of immune reconstitution phenomena.

R F MILLER

P J SHAW

I G WILLIAMS

Department of Sexually Transmitted Diseases, Royal Free and University College Medical School, Mortimer Market Centre, Mortimer Market, off Capper Street, London WC1E 6AU

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Accepted for publication 26 November 1999

BOOK REVIEWS

Common Gynaecological Problems. Ed by Patricia Wilson. Pp 312; Price £24.95. Oxford: Blackwell Science, 1999. ISBN 0-632-05174-4.

A book with a title such as this one makes it difficult for the author to decide what to exclude. This book certainly fulfils its major objective of providing an easy reference manual for the diagnosis and management of common gynaecological conditions. It deals with almost all the gynaecological conditions that could be encountered in the community and the common gynaecological problems in hospital medicine. Overall, the topics covered are well presented with special points highlighted.